the beginning of the tumor. This must be taken to lie in all probability in the peripheral layers of the substantia compacta.

We can acquire more information by turning to the sagittal section of a fusiform tumor of the femur sketched in Fig. 19. Our inspection reveals two significant facts—first, the evidence of the osteoplastic activity of the periosteum, and second, the intact condition of the substantia compacta. How these are to be interpreted shall form a topic for discussion when the point of origin of the sarcomas shall receive special consideration.

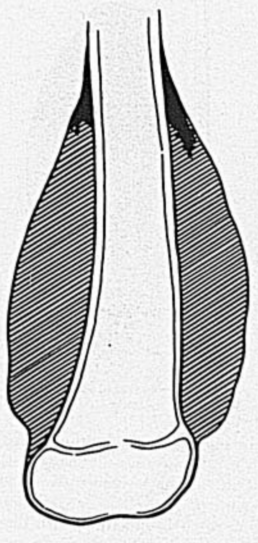


FIG. 19.—Subperiosteal sarcoma of the femur.

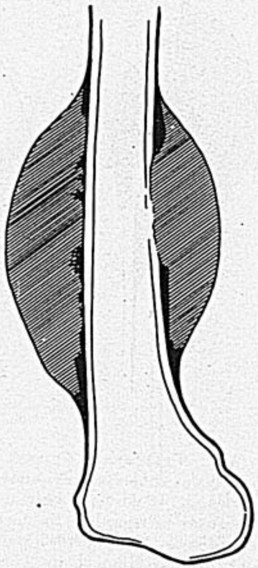
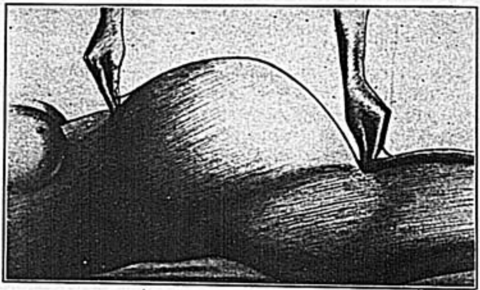


FIG. 20.—Peripheral sarcoma of the femur.

Perhaps somewhat more comprehensible from the standpoint of genesis is the peripheral sarcoma shown in Fig. 20, in which there is a tumor encircling the shaft. Toward either pole we can detect the strata of periosteal bone deposit, these becoming more elevated as they approach the centre of the neoplasm. Then there is a sudden interruption in the continuity of these lamellæ, and we see a shallow excavation at the posterior part of the shaft. It is at this point that

After the sixth month this rule is extraordinarily exact, and is most useful in determining the date of labor and the size of the fœtus, when the date of the last menstruation has been forgotten. It has been in use in my hands since 1904, and I have had good reports of it from many obstetricians, including some of my German confrères. Hamilton has reported it to me to be of great use in asylum practice in insane pregnant women who are not able to give a connected history of menstruation.



Author's rule: The duration of the pregnancy in lunar months equals the height of the uterus in centimeters divided by 3.5.

It may be said that 35 cm. is the usual height of the uterus at full term with a fœtus of 3300 grams. For every centimeter of height above this measurement approximately 200 grams should be added to the weight of the fœtus. Thus, a uterus measuring 37 cm. would contain a fœtus weighing 3700 grams. The measurements are more exact below 35 centimeters, than above that height.

The so-called "sinking" of the fœtus in the last two weeks of pregnancy causes but little error in the measurement, as the head, when the patient is recumbent, rides upward on the pelvic bones and the sinking is not a factor. "Sinking" in my experience is not common in primiparæ, and its supposed presence is often due to the stretching of the abdominal muscles and not to descent of the head into the pelvis. The fundus thus comes lower in the erect position, and no diminution of the fundal height is noted in the recumbent. This is well shown in Hirst's[13] photographs of women at various periods of pregnancy. "Sinking" does, however, in multiparæ sometimes complicate the measurement, but not often. Hydramnios also causes but small error, as the excess of liquid does not affect the fundus, but the body of the uterus, leaving the height of the fundus to be determined by the occipitococcygeal measurement and the size of the fœtus.

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[13] Text-book of Obstetrics.

The rule gives the most exact means at hand of prognosticating the date of labor. No rule can be exact when dealing with such an uncertain quantity as the duration of pregnancy, save that in the majority of cases an average-sized baby is born at the average time.

Thus, if the fundus measures 26 centimeters from the symphysis, the duration of pregnancy is 26 divided by 3.5 or $7\frac{3}{7}$ lunar months, and the patient has $2\frac{4}{7}$ lunar months to go to term, or ten weeks and two days.

This rule, combined with the estimation of pregnancy by reckoning from the last menstruation, gives a fairly exact determination of the probable date of labor.

While the rule is useful for the determination of the date of labor, it is still more useful for the determination of the size of the fœtus, with a view to induction of labor for contracted pelvis or other cause. When the fundal measurement is at or near 35 cm., I never hesitate to induce labor when indicated, knowing that there is a fœtus of the average weight of about 3300 grams and capable of standing instrumental delivery and not liable to die from prematurity.

When used for the purpose of estimating the size of the child in contracted pelvic or other conditions, it should be used in conjunction with the other methods of measuring the head, etc., as noted in my previous paper on this subject.

FURTHER STUDIES OF SARCOMA OF BONE.[1]

By Leo Buerger, M.D.,
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With the knowledge of the structural changes in bone that can be gained by means of the Röntgen rays, and with the aid of the newer viewpoints in the histopathology of sarcoma, we are in a position today to acquire a much more throrough and comprehensive conception of the method of growth of sarcomas of bone than has been possible in the past. In a previous publication,[2] giving observations on 20 bone sarcomas, certain characteristic features were described and their importance in diagnosis was pointed out; the utility of adhering to the histological classification of Borst was demonstrated and a tentative grouping into seven types, each distinguished by certain definite gross morphological criteria, was suggested as being at least of some temporary value. Since that time my own experience has been enriched by a number of additional clinical cases; two more specimens of sarcoma of the long bones have been studied, as well as two cases of multiple myeloma. The very excellent account of Rumpel's[3] radiographic findings in bone sarcoma has been compared with the data acquired by x-ray and anatomical investigation of my own material, so that I am able to give a review based on some 30 clinical cases and 46 specimens. Whereas it was possible to give the histopathology of my own material a thorough consideration, the available information on this point in the case of Rumpel's sarcomas is too scant to warrant a comparative exposition of this phase of the subject. I shall, therefore, confine myself to the conclusions that the study of the gross structural changes in 46 cases of bone sarcoma may permit.

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[1] From the Department of Surgical Pathology of the Mt. Sinai Hospital, New York.

[2] Surg., Gyn., and Obstet., October, 1909, 431 to 461.

Ribbert,[4] in his admirable inquiry into the development of these tumors, has shown that many of our old notions, particularly in regard to genesis, ought to be abandoned. My own experiences are in the main in accord with his, especially in so far as they concern the probable seat of origin; but they are at variance with his conclusion in the matter of the site of inception of the so-called "peripheral" growths. I shall revert to this later on.

Let me briefly epitomize some of the distinguishing attributes of the bone sarcomas, so that an understanding of individual characteristics may be facilitated.

From a consideration of the many data before us we are led to an endorsement of a view recently advanced, namely, that true "periosteal" sarcomas are probably so rare as to be negligible, and that the bone sarcomas find their incipiency in a focus somewhere within the confines of the osseous tissue or perhaps just under the periosteal covering. Leaving aside the arguments in favor of this opinion for the present, let us assume the beginnings somewhere within the peripheral layers of the substantia corticalis, in the medulla, in the corticalis itself, or in the spongy bone, and the following structural changes will be seen to take place. There will be a disintegration and disappearance of the osseous tissue and marrow *pari passu* with the enlargement of the sarcoma, although in the case of certain varieties, new bone formation either on the part of the tumor or on the part of the irritated normal bone elements may occur. The method of advance will vary in the way to be described later. Here it suffices to note that sooner or later the heterotypical cells will have destroyed some of the cortex and have found their way under the periosteum, which they will lift off as they multiply. By virtue of this detachment (and possibly also because of the stimulation of the tumor cells themselves) proliferative changes are induced in the bone covering, and new osseous lamellæ are laid down. In the earliest stages this forms a bony thickening over the cortex indistinguishable from an inflammatory periosteal deposit. Soon, however, the neoplasm tends to encircle the bone, carrying the periosteum with it. A fusiform tumefaction results, the ends of the spindle presenting on section characteristic wedges or triangular areas of periosteal bone. Later, the periosteum too is broken, and its remnants with attached portions of new-formed bone may become included in the tumor.

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[3] Ueber Geschwülste und entzündliche Erkrankungen der Knochen im Röntgenbild Hamburg. 1908.

[4] Geschwulstlehre, 1904; and Beiträge zur Entstehung der Geschwülste, 1906.

In this short characterization it has been deemed advisable to call attention only to a very few but essential facts, namely, the origin in the bone tissue or marrow, the destruction of the bone, and the role of the periosteum: for these will aid in the detailed study now to follow.

Turning our attention to the comparative study of our own 22 specimens and of the 24 described by Rumpel, we will find that the tentative grouping proposed by me is also of service in classifying the tumors portrayed in that author's Atlas of Riadograms.[5] The following is the grouping under discussion:

A. CENTRAL (ENDOSTEAL) TUMORS.

1. Expansive of the bone ends.
2. Expansive of the shaft.
3. Diffusely infiltrating (non-expansive).
4. Destructive, dissolutive (non-expansive).
5. Sclerosing (calcifying).
6. Perforative (non-sclerosing).

B. PERIPHERAL TUMORS.

7. Superiosteal and true peripheral.

I. Expansive Tumors of the Ends of Long Bones. Exquisite examples of this form are the giant-cell sarcomas usually encountered in the lower end of the femur and upper end of the tibia and humerus. They begin as a rule in the spongiosa of the diaphysis, not far from the epiphyseal line and exhibit a marked tendency to bring about complete disintegration of the osseous tissue. By virtue of necrosis, liability to hemorrhage, and regressive metamorphosis, larger or smaller cavities are produced. As for their "expansive nature" (Figs. 1 and 2), this needs qualification, since it is often more apparent than real. Although a moderate amount of enlargement of the extremity of the bone may be produced by the tumor growth even before the investing cortical sheath has been invaded, this is but meagre when compared with the dilatation incident to the later destructive changes. Complete arrosion of the corticalis is the result when the tumor attains considerable size. The periosteum, however, may remain as an encapsulating vestment, and hand in hand with its recession from the surface there occurs the development of new bone, so that a new shell of thin osseous lamellæ may form the chief covering of the neoplasm. The eggshell or ping-pong ball[*] crackle, therefore, may be elicited by pressure on either rarefied cortex or new formed periosteal bone.

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[5] Loc. cit.

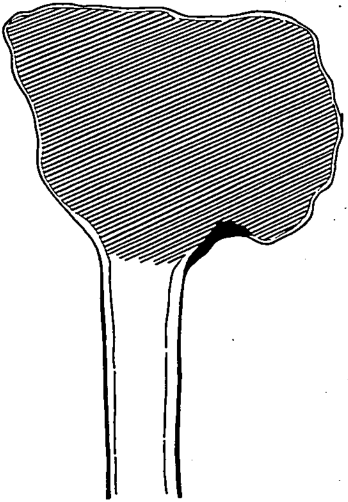

FIG. 1.—Giant-cell sarcoma of the tibia.

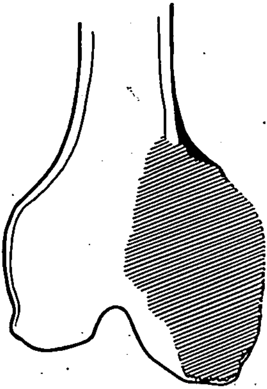

FIG. 2.—Giant-cell sarcoma of the femur.

We may regard the illustrations (Figs. 1 and 2) as depicting types of this variety. The first, a giant-cell sarcoma of the tibia, belongs to my series, and the second, a giant-cell sarcoma of the femur, to Rumpel. Besides the features already mentioned in a general way, we should note the following: The triangular area of subperiosteal bone, the fact that the articular surface is intact, and the evidence of threatened complete destruction of corticalis. Such evidence is seen in Fig. 2, where there is absence of cortex over the upper half of the tumor, and substitution by delicate new osseous plates. Here the tumor has evidently escaped from its bony confines and is partly extra-osteal, although limited, for the most part, by intact periosteum. Similar growths occur in the upper end of the humerus, although here the dilatation of bone is not clinically as apparent as in the case of the femur and tibia.

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[*] Suggested by Dr. Bloodgood.

It is these tumors that may be easily confused with bone cysts. If we remember that cysts do not perforate, but cause rarefaction and expansion of the cortex, that they are surrounded by a smooth bony wall, and that reactive bone proliferation either in the bone itself or on the part of the periosteum does not occur with them, we shall have at our disposal a sufficient number of data for differential diagnosis.

That other forms of cystic sarcoma (myxosarcoma) and more malignant varieties (osteoblastosarcoma) may also belong to this group, I have already mentioned elsewhere.

II. Expansive Tumors of the Shaft. A brief mention of the occurrence of expansive tumors in the diaphysis will be enough to acquaint us with this type. In my series there was a chondrosarcoma of a metacarpal bone and one of the femur. In Rumpel's list there was a sarcoma developing in an enchondroma of the humerus which had much in common with my specimen of the femur. The same picture of bone destruction that is seen in the last type occurs here. The available material is hardly adequate to allow of formulating any definite rules as to what radiographic pictures ought to be expected. It is evident, however, from the data at hand, that some difficulty in the differentiation between benign enchondroma and chondrosarcoma can hardly be avoided. A reference to Fig. 3 illustrates how the picture characteristic of enchondroma may be produced by a cartilaginous growth whose tissue showed, microscopically, unmistakable traits of malignancy, although a true malignant nature could not be clinically proved in this case. In the enchondromas (Fig. 4) we not rarely encounter multiple tumors that cause disappearance of the osseous tissue, leaving a structure which in the *x*-ray picture has a speckled and rarefied appearance. The absence of periosteal deposits is one of the most important aids in distinguishing them from many of the types of central sarcoma of bone.

Two other conditions that may stimulate the central expansive sarcomas of the shaft may be mentioned—bone cysts, and cyst formation in so-called "osteitis fibrosa." The enchondromas and bone cysts both from the clinical standpoint and from the skiagrams may be practically identical, so much so that a close relationship seems not unlikely. Indeed, the bone cysts are in all probability the end result of a number of different types of endosteal disease. It is not my purpose here to enter into a discussion of the differential diagnosis between these conditions and sarcoma, inasmuch as this can be best treated from a consideration of a large number of radiographic plates. Here it may suffice to call attention to the following points: In bone cysts, fusiform or pyriform bone expansion, rarefaction of the substantia compacta with the greatest absorption at the equator of the tumefaction, and a distinct, regular line of demarcation between cyst and bone tissue, are characteristic; whereas irregular expansion, perforation, extra-osteal masses, periosteal bone deposits, and irregular contour are more apt to be among the qualities of the malignant growths.

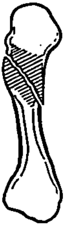

FIG. 3.—Chondrosarcoma of a metacarpal bone.

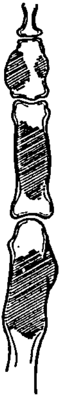

FIG. 4.—Enchondromas of the phalanges and metacarpal bones.

III. DIFFUSE INFILTRATING (NON-EXPANSIVE) TUMORS. Quite a contrast to the last described series is presented by those sarcomas that probably originate in the innermost layers of the substantia compacta or in the medullary substance, when they develop in the shaft, and probably begin in the spongiosa when they belong to the extremity of a bone. Here expansion of the corticalis is missed, and we find a displacement of osseous tissue and marrow, with marked tendency to intramedullary extension. We are often surprised, on section of the bone, to see how far in the longitudinal direction the tumor growth has advanced. Fig. 5 shows the diffuse involvement of the head and shaft of the femur in one of my series of four specimens of this type and illustrates the tendency to pathological fracture, which may be one of the first indications of the presence of these growths. In Rumpel's series I could find but one example—a round-cell sarcoma of the ulna, which is depicted in

Fig. 6. Here we see the wide endosteal extension with relatively little destruction of substantia compacta, and will note the small wedge of bone deposits from the periosteum where that has been displaced by extra-osteal growth. Of the four examples in my collection, there were two of the upper end of the femur, one of the shaft of the tibia, and one of the shaft of the humerus (Fig. 7).

This type ought to be recognized as of clinical importance, inasmuch as an early diagnosis will hardly be made. For the endosteal tumor may have already replaced a large portion of the medulla and innermost layers of corticalis, or may have caused pathological fracture, long before a suspicion of neoplasm is entertained. Thus, the diagnosis was made after spontaneous fracture in one case, ofter osteotomy in two cases, and after careful radiographic examination and exploratory incision in a fourth case. The patients do not usually consult us until after considerable bone destruction has taken place. An illustration of a diffusely growing round-cell sarcoma of the humerus may be of interest in representing this type (Fig. 7).

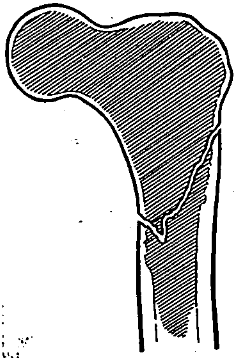

FIG. 5.—Fibrosarcoma of the femur.

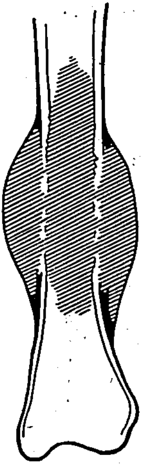

FIG. 6.—Diffuse infiltrating sarcoma of the ulna.

IV. DESTRUCTIVE, DISSOLUTIVE TUMORS. When a sarcoma becomes so highly destructive that there is rapid dissolution of the whole region surrounding its point of origin, and when there is neither a tendency to expansion nor to demarcation of the neoplasm, we have a growth which may be grouped under "destructive" or "dissolutive tumors." I have seen some half-dozen such sarcomas of the upper end of the fibula, one of which is depicted in Fig. 8. The invasion of the surrounding soft parts occurs early and the tumors usually pursue a very malignant course. Rumpel's series affords two such specimens, one of the upper end of the tibia, another of the upper end of the fibula. Fig. 9, a diagrammatic drawing from a sagittal radiogram, illustrates the manner of growth in a case in which the upper end of the tibia is completely destroyed and substituted by a very large tumor. Where the tumor merges with the intact shaft are seen some evidences of bone proliferation. The periosteal bone can be traced upward in the form of two irregular lines which represent what is left of a new cortex made of periosteal bone. Here we see a demonstration both of the reactive periosteal osteoplasia and of the fact that the new-formed bone will in its turn be disintegrated by the advancing neoplasm.

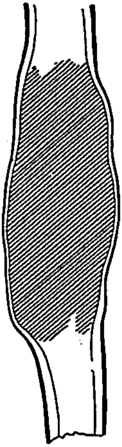

FIG. 7.—Diffuse infiltrating round-cell sarcoma of the shaft of the humerus.

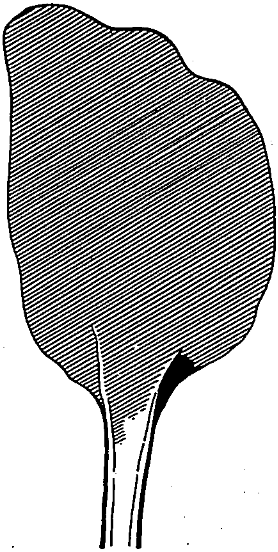

FIG. 8.—Dissolutive (round-cell) sarcoma of the upper end of the fibula.

V. Sclerosing, Calcifying (Ossifying) Tumors. Perhaps one of the most interesting varieties of bone sarcoma is that in which an osteoid or chondroid matrix is elaborated. Hand in hand with this higher differentiation into a typical bone- and cartilage-producing mesoblastic tissue goes a tendency to sclerosis, in the sense that larger or smaller areas of the tumor are converted into stony masses. This is accomplished either by a sort of lawless calcification or by the deposition of lime in the osteoid matrix and the consequent production of a typical bone. Having already had six examples when writing my last paper[7] (three of the femur, two of the tibia, and one of a rib), the histological and gross characteristics were sufficiently dealt with at that time. Here it may be permissible, for the sake of clearness, to repeat the following: That calcification and bone formation may be both intra- and extra-osteal; and that the shaft in which such a tumor originates may be converted into a dense petrified mass with such fusion of the remnants of corticalis and intra-osteal tumor, that it is impossible to tell macroscopically where the tumor ends. This remarkable feature of deposition of lime gives a distinctive stamp to the radiograms and is therefore of

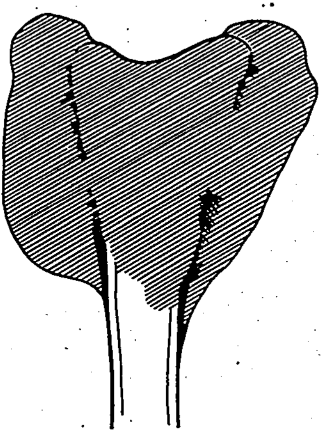

Fig. 9.—Dissolutive, round-cell sarcoma of the upper end of the tibia.

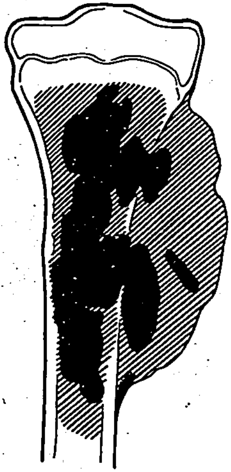

Fig. 10.—Sclerosing sarcoma (osteoid) of the tibia.

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[7] Loc. cit.

service in diagnosis. Thus, Fig. 10, depicting an osteoid sarcoma of the tibia, represents diagrammatically the endosteal hardening, dense masses of lime filling the bone and also forming a core in the soft extra-osteal tumor, so that in some of the longitudinal sections an imperceptible transition from calcified tumor into altered cortex can be demonstrated.

When these tumors make their way out under the periosteum, they give rise to the same bone proliferation seen in the other varieties, except that we must expect an additional osteogenetic process in the basal portions of the tumor, namely, that which rides on the shaft of the bone. In the extra-osteal tumor-spindle the typical radiate arrangement of bony spicules is sometimes developed, especially in the chondroid varieties. That this feature is not a distinguishing characteristic of the "periosteal" tumors—as was formerly supposed—but belongs rather to those central tumors that stimulate the periosteal variety will become evident from our analysis.

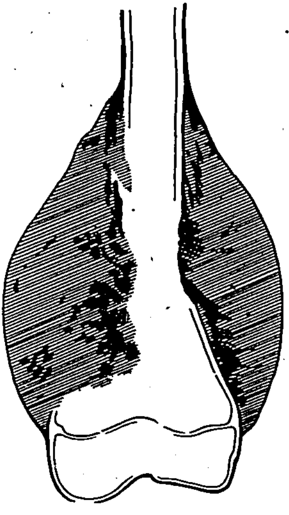

FIG. 11.—Sclerosing sarcoma of the femur.

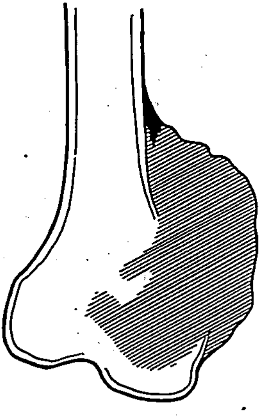

FIG. 12.—Perforative sarcoma of the femur.

In my own collection, the calcifying and ossifying type was represented only by the osteoid and chondroid varieties, although in one of the round- and spindle-cell tumors, a fair amount of irritative bone proliferation—on the part of the normal bone—was rather striking. Rumpel pictures marked sclerosis in a spindle-cell sarcoma. Bearing in mind the fact that the sarcomas with specialized intercellular substance, such as the chondroid and osteoid, may, in the course of their growth, revert to immature, low types (spindle- and round-cell forms), and that we not infrequently find soft immature sarcoma in the metastases and extra-osteal masses, it seems possible that osteoid or chondroid substance may be overlooked if the central portions—or the oldest parts—are not subjected to histological examination. Be that as it may, the radiogram from which Fig. 11 was drawn shows not only the irregular areas of lime infiltration in the extra-osteal tumor, but also the typical spicules and the dense nature of the shaft itself. Whether sclerosis of the nature described can occur in the other forms of sarcoma, I am unable to say from the data at my disposal.

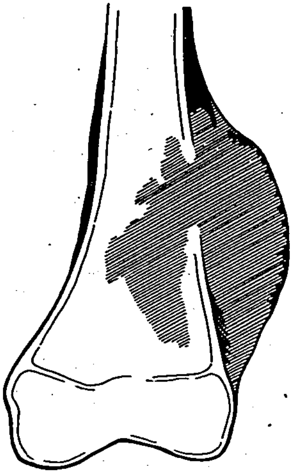

Fig. 13.—Perforative sarcoma of the femur.

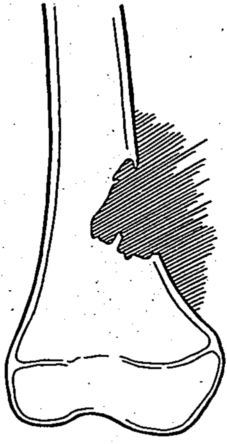

Fig. 14.—Perforative sarcoma of the femur

VI. Perforative Endosteal Tumors. Instead of a large central focus causing expansion, or diffuse endosteal infiltration, or extensive bone destruction, or sclerosis, we may see a totally different picture, namely, a small intra-osteal mass causing early perforation. Such a growth is exemplified by the specimen in Fig. 12, portraying a non-calcifying soft osteoid tumor of the femur. In the analysis of Rumpel's sarcomas, it was found that this type is not at all unusual. Seven of his tumors could be placed into this class, 2 of the tibia, 2 of the humerus, and 3 of the femur. Diagrammatic reproductions of the 3 tumors of the femur are to be seen in Figs. 13, 14, and 15. In all of them the sarcoma had its origin in the spongiosa of the diaphysis, and there was a relatively small endosteal focus with large extra-osteal masses. The other features already sufficiently described can be readily detected by a glance at the illustrations.

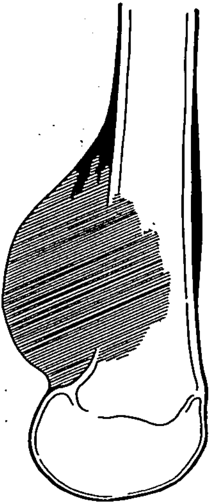

Fig. 15.—Perforative sarcoma of the femur.

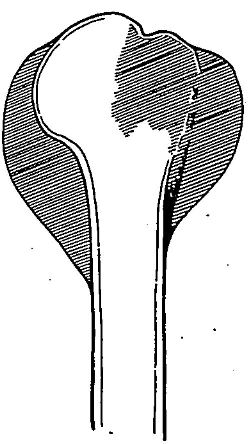

Fig. 16.—Perforative sarcoma of the humerus.

We need hardly emphasize the difficulty and, in most cases, the impossibility of diagnosticating this type of sarcoma before perforation has taken place. When the humerus is affected, the recognition of the condition, even with radiograms, may tax our powers to a still greater degree. The meagre changes in the bone may go with considerable tumefaction of the soft parts and the diagnosis of arthritis may be made. A glance at Fig. 16 will give us a notion as to the lesion in one case.

Another example of this form is shown in Fig. 17, in which the outer tuberosity of the tibia is occupied by a small tumor which has already made its way out of the bone. Such a tumor must be differentiated from a bone cyst, osteomyelitis, or bone abscess, and from a central gumma. The evidences of a lesion outside of the bone, the irregular contour of the endosteal area of bone absorption, and the periosteal deposits are sufficient to rule out bone cyst. Nor do we meet with the peculiar circumferential osteophytes in central gumma or in abscess.

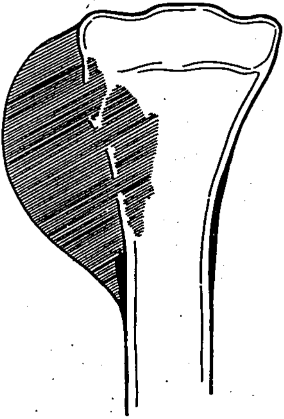

Fig. 17.—Perforative sarcoma of the tibia.

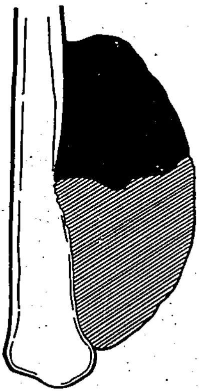

Fig. 18.—Subperiosteal sarcoma of the femur.

VII. Peripheral Tumors. Having had but one tumor whose origin was either "subperiosteal" or in the outermost lamellæ of the cortex, it would have been audacious to have expressed myself decisively in regard to the moot point of periosteal or peripheral origin in my first paper.[8] Comparing my own specimen with those described by Rumpel, the conclusion is inevitable that a certain percentage of the bone sarcomas do belong to this class, and that in a series of eight tumors, at least, the periosteum, although affected by the growth, is not the seat of its inception. Turning our attention to Fig. 18, showing a large osteosarcoma[9] of the femur, the lack of bone disintegration will be noted as a striking feature. Indeed, there is only very superficial arrosion of the shaft, at a point marking the beginning of the tumor. This must be taken to lie in all probability in the peripheral layers of the substantia compacta.

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[8] Loc. cit.

[9] By osteosarcoma is meant not a sarcoma in bone, but a bone-forming sarcoma.

We can acquire more information by turning to the sagittal section of a fusiform tumor of the femur sketched in Fig. 19. Our inspection reveals two significant facts—first, the evidence of the osteoplastic activity of the periosteum, and second, the intact condition of the substantia compacta. How these are to be interpreted shall form a topic for discussion when the point of origin of the sarcomas shall receive special consideration.

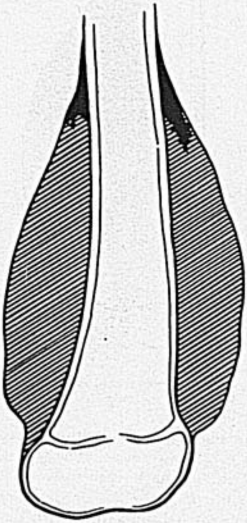

Fig. 19.—Subperiosteal sarcoma of the femur.

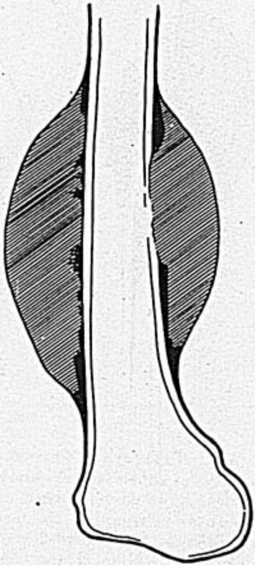

Fig. 20.—Peripheral sarcoma of the femur.

Perhaps somewhat more comprehensible from the standpoint of genesis is the peripheral sarcoma shown in Fig. 20, in which there is a tumor encircling the shaft. Toward either pole we can detect the strata of periosteal bone deposit, these becoming more elevated as they approach the centre of the neoplasm. Then there is a sudden interruption in the continuity of these lamellæ, and we see a shallow excavation at the posterior part of the shaft. It is at this point that we must seek the point of origin. We need hardly add many more examples. One more will suffice to give us a definite notion both as to origin, development, and as to what diagnostic points can be expected from the radiograms. How little the shadow pictures may show is apparent when we look at Fig. 21, a diagrammatic representation of a skiagram of the specimen. In the x-ray of the limb there was nothing but the slight indication of circumferential periosteal osteoplasia, and a roughened line indicating surface arrosion. These two features are well shown in the figure. Thus, the characteristic marks identifying such peripheral sarcomas are periosteal bone deposit, eroded bone, and the presence of an extra-osteal mass.

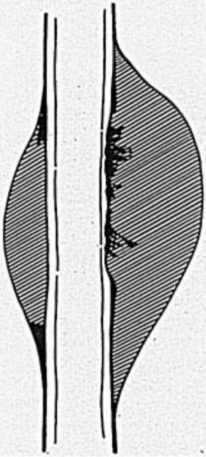

FIG. 21.—Peripheral sarcoma of the femur.

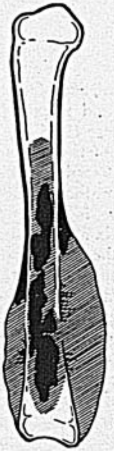

FIG. 22.—Sclerosing sarcoma of the tibia.

Is it always possible to distinguish in the skiagrams the lesions of sarcomas of small extent from those that belong to a traumatic or inflammatory periostitis? The differential diagnosis may be difficult, for the shadow pictures may show only periosteal deposits without appreciable changes in the bone itself. The circumferential type of bone deposit usually speaks in favor of neoplasm, whereas in the traumatic periostitis we are more apt to find irregular, and possibly multiple patches of bone deposit. However, it is clear that even in sarcoma the very earliest periosteal lesion will not have encircled the shaft, and this early neoplastic lesion may, therefore, be difficult or impossible of recognition.

GENERAL CONSIDERATIONS. Having thus outlined the characteristics of forty-six bone sarcomas, and having grouped them according to gross morphological traits, it may be well to gain a broader impression of the subject by tabulating both our own specimens and those of Rumpel and by giving the subject of classification and point of origin more critical consideration. The distribution was as follows:

	Rumpel.[10]	Buerger.[11]	Total
Femur	8	11	19
Tibia	5	4	9
Humerus	6	1	7
Fibula	2	4	6
Metacarpal	1	1	2
Radius	1	0	1
Ulna	1	0	1
Rib	0	1	1
Total	24	22	46

In order of frequency we would have femur, tibia, humerus, fibula, and metacarpal. Reinhardt[12] collected 54 cases in which there were 19 of the tibia, 18 of the femur, 13 of the humerus, and 2 each of the fibula and radius. Müller,[13] in reviewing 209 sarcomas, found the tibia affected 77 times, the femur 70, the humerus 38, the radius 11, the ulna 6, and the fibula 8. The tibia and femur, therefore, take the first place, followed by the humerus, and then by the fibula and radius.

Has the comparative study of this larger number of bone sarcomas given impressions that would tend to strengthen notions already obtained through our previous investigation? I must answer in the affirmative. A suspicion has arisen that perhaps certain of the groups do not possess any essential properties with which the gross alterations are inseparably linked, and that the changes of bone architecture depend for the most part on variations in the site of origin of the neoplasm. However this may be, the establishment of the existence of important widely variant forms is not nullified; and we must concede that, although the assumption as to the rôle of origin may be true in the case of the "border-line" tumors, most of the neoplasms possess a sufficient number of distinctive qualities to allow of the grouping adopted by us. Tabulating Rumpel's tumors and my own, we find the 46 sarcomas grouped as follows:

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[10] Includes only those critically studied by me, the data being sufficient.

[11] The multiple myelomas are not added. Only specimens which were studied anatomically are included in my list.

[12] Deutsche Zeitsch. f. Chir., 1897, xlvii, 523.

[13] Inaug. Diss., Kiel, 1904.

	Rumpel.	Buerger.
I. *Expansive of the Extremities of Bones.*		
Femur	2	3
Humerus	1	
II. *Expansive of the Shaft.*		
Humerus	2	
Femur	–	1
Metacarpal	–	1
III. *Diffuse Infiltrating.*		
Femur	–	2
Tibia	–	1
Humerus	–	1
Ulna	1	
IV. *Dissolutive (destructive).*		
Fibula	1	3
Femur	–	1
Tibia	1	1
V. *Sclerosing, ossifying, calcifying.*		
Femur	1	3
Tibia	1	2
Rib	–	1
VI. *Perforative endosteal.*		
Femur	3	1
Humerus	2	
Tibia	2	
VII. *Peripheral.*		
A. Subperiosteal[14] (shaft)	1	1
B. True peripheral:		
Femur (shaft)	1	
Humerus (shaft)	1	
Radius (shaft)	1	
Tibia (shaft)	1	
Fibula (shaft)	1	
Metacarpal (shaft)	1	
Total	24	22

Rather striking is the large number of perforative endosteal tumors, of which I have had but one example, and of which Rumpel —if my interpretation of his radiographic findings is correct—has had 7. This augmentation in the number of examples of a type which had to be separated from the rest in my series (although there was only one) makes it certain that this variety is to be reckoned with. None the less remarkable, and a circumstance which has somewhat altered my former concept, is the large proportion of peripheral sarcomas—7 out of 24 as compared with 1 out of 22 in my list.

Let us set aside the question of the fitness of a special classification as well as the critique of the one proposed until we shall have discussed more fully the manner of growth of the bone sarcomas and their site of origin. We owe much of our knowledge of these two phases of the subject to the admirable work of Ribbert. He was one of the first to emphasize the endosteal origin of these neoplasms, and to recognize the role of the periosteum as being active rather in the sense of producing new bone than in the sense of participating in the production of the tumor itself.

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[14] With very slight or practically no erosion of the corticalis,

In the bony tissue the sarcoma elements soon cause a disappearance of the normal structure, with a rapid resorptive process, the bone melting away, as it were, before the advancing cells of the neoplasm. The bone absorption seems, for the most part, to be the result of the direct action of the tumor cells, although we not infrequently see osteoclasts engaged in a similar process. At the same time, an interesting phenomenon may occur, namely, the deposition of new matrix upon the very bone tissue that is undergoing destruction. Such osteoplasia needs careful interpretation, since it may either be a normal reactive homeotypical bone proliferation, such as is the rule in syphilis and chronic inflammatory processes, or it may be heterotypical or atypical, the result of the activities of bone-forming, osteoblastic sarcoma elements. The former type is usually not sufficient in amount to produce architectural changes that need special consideration from the standpoint of diagnosis in the skiagrams; the latter, on the other hand (belonging par excellence to the osteoid, chondroid, and true osteosarcomas), is responsible for the striking lime and bone metamorphosis which has already received special mention elsewhere. In one of my cases of the dissolutive type of sarcoma of the fibula the "normal" type of bone formation was fairly striking. Indeed, it did much toward preserving the general contour of the bone, just as is the case with the more solid, dense, calcified endosteal sarcomas of the osteoid variety.

By virtue of advance through Haversian canals or by direct extension through places in the substantia corticalis that have undergone complete absorption, the neoplasm finds its way under the perisoteum. The latter becomes separated from its bony support, first over a small area, by reason of the progress of the tumor, and later because of hemorrhages between the periosteum and the surface of the bone. It is now that the peculiar phenomenon of new bone formation under and by the periosteum takes place. A study of the skiagrams of gross specimens reveals the interesting fact that the bone deposition occurs in two forms—either as flat stratified lamellæ, disposed parallel with the long axis and surrounding the bone, or in the shape of fine trabeculæ that have a radiate arrangement, lying perpendicularly to the shaft. Both of these varieties can frequently be found in the same specimen. Although my studies of this particular phenomenon cannot be regarded as sufficiently thorough to warrant a decided opinion, it seems to me now that the following explanation will hold good: The flat lamellæ represent, in part, bony deposits that are the result of bone proliferation from the peripheral layers of the corticalis, and, in part, the proliferation that occurs when the periosteum has suffered but minute separation from the bone. Such separation is not confined to the immediate variety of the perforating sarcoma tissue, but extends for a considerable distance up and down the shaft and in a circumferential direction. Thus, the first consequence of cortical perforation will be the growth of a thin plate of bone upon the shaft. As the tumor enlarges, a tendency to form a lenticular-shaped growth becomes manifest. The periosteal elevation may occur at intervals, depending upon the subperiosteal tension, and each fresh act of separation will be followed by a new accession of lamellar bone growth. Simultaneous with this another mode of periosteal activity is evidenced in the growth of bone trabecula from the periphery toward and perpendicular to the bone. Such radiate disposition is particularly shown in the radiograms, and according to Ribbert depends on the fact that the perforating vessels have been pulled out in that direction and must to a certain extent determine the direction of the growth. Be that as it may, we need but note that where the bone covering has suffered any considerable displacement, there the radiate arrangement is rarely missed, whereas where the periosteum is separated but a few millimeters or less than a millimeter, the lamellar structure is more apparent

At the poles of the extra-osteal tumor, where the transition between normal and uplifted periosteum must be sought, a characteristic wedge (in section) or conical shell of bone is regularly seen. The importance of recognizing this structural marking, both in diagnosis and in the estimation of the site of origin, will receive attention later on.

I have thus far called special attention to the proliferative phenomena induced by the extra-osteal growth, It can readily be appreciated, however, that the new-formed periosteal bone will in its turn suffer the same destruction which had already befallen the existing osseous tissue. As a result, the central parts of the cortex of extra-osteal spindle will become deprived of its bony covering. A fibrous capsule, the last remnant of periosteum may be left, or rupture of this may occur, in consequence of which rapid growth into the soft parts or around the periosteum occurs. The periosteum then lies in the tumor mass. This explains the occasional inclusion of periosteum, with its adherent bone, in parts of extra-osteal tumors.

From this description it is evident that, if it is possible to determine the presence of intact periosteum, or to detect evidence of bone proliferation in it, or to trace its dislodgement in the manner suggested, we must be prepared to controvert the opinion that such perisoteum contains in it the nucleus of inception of the neoplasm. We must rather seek a focus under the periosteum or in the bone itself.

After this slight digression into the question of origin of the sarcoma, I shall close the chapter on mode of growth by calling attention to the common varieties of extra-osteal tumor.

The fusiform type is the ordinary variety. This form may give us—as shall be seen later—no indication as to where the beginnings of the tumor lie. Whereas, the true spindle, with tapering ends, may be found when the tumor is limited to the shaft, a blunt, and even fungiform overhang may mark its limit at the extremity of a long bone. The absence of the typical periosteal bone wedge may be ascribed to the dense adhesion of the membrane at the epiphyseal region. Besides this, we need only mention the nodular lobules of extra-osteal metastases occasionally seen, and the irregular forms that belong to the stage of periosteal destruction and infiltration of the soft parts.

THE ORIGIN OF THE BONE SARCOMAS. In the light of what has been said we can now better understand the presumptive point of origin of the tumors belonging to our series. Let me therefore analyze the various groups in the sequence previously given. Having done this, I shall be able to proceed to the critique of the proposed classification, with a more complete appreciation of its merits and shortcomings.

Fortified with the knowledge of the role played by the periosteum in the development of the bone sarcomas, we must abandon the old conception of division into myelogenous and periosteal varieties. Ribbert recognizes central and peripheral forms. The former originate in most instances in the substantia spongiosa of the diaphysis, although they may grow from any point that lies within the outermost layers of the corticalis, and the latter are derived from the outer layers of the periosteal bone. Ribbert would have even these so-called peripheral tumors start near one end of the diaphysis, and creep up or down the shaft. I shall be able to show that at least this latter view is not in accordance with the data at our disposal.

In establishing the location of the beginnings of the malignant tumors, we must bear in mind a few fundamental facts. A tumor will grow with equal rapidity in all directions if the resistance offered on all sides is approximately the same. It is just here in the sarcomas of bone that we encounter several difficulties in the proper interpretation of the point at issue. These are the differences of rate of proliferation in bone and in soft parts, and the limiting powers of the substantia compacta, of the articular cartilage, and the periosteum. Thus, we cannot off hand place the site of initial growth in the centre of the gross product.

Turning to our series, the expansive tumors of the ends of the long bones will be found to arise from a point somewhere in spongy bone of the end of the diaphysis. Beginning for the most part in the upper end of the tibia or lower end of the femur, the equal enlargement will leave no doubt as to the correctness of the assumption that they are central in origin.

When we come to consider the expansive tumors of the shaft and the diffuse infiltrating type, we meet with a more intricate problem.

Here we may assume either a focus in the marrow, in the innermost layers of the substantia compacta, or in the spongiosa of the diaphysis. In view of the nature of the histological types (giant-cell and chondral), it seems most likely that an osseous rather than a myelogenous focus is their starting point.

The dissolutive form also arises in all probability in the spongiosa of the diaphysis. The equal destruction of the bone ends is sufficient testimony in favor of this view.

Perhaps none of the tumors afford as much food for investigation as the sclerosing and ossifying sarcomas, when they form fusiform bone-encasing spindles, or when they cause that fusion of calcified tumor and bone that makes the identificaton of neoplasm so difficult. Here we meet with growths that show both endosteal and extra-osteal proliferation. The focus in the bone may be smaller than the mass without. In the interior of the bone there is usually calcification, and often greater in extent than that which forms the petrified core of the extra-osteal masses. In these forms the dense nature of the intra-osteal focus and the evidences of cortical perforation and the separation of the periosteum leave little doubt that they are endosteal in origin. The beginnings were referable to a site in the spongiosa of the diaphysis in 5 of my specimens and to spongiosa of the rib in a sixth.

Exquisite examples of endosteal origin are given us in the perforative type, in which the central focus is usually much smaller than the extra-osteal mass. If we glance at Figs. 12 to 17, inclusive, we shall not escape entertaining the suspicion that perhaps the small intra-osteal tumor is but an expression of invasion from without. Here we are not aided in our estimation of the age of the newgrowth by such a phenomenon as calcification. When we study the relative size of extra- and intra-osteal neoplasms, and keep in mind the greater rapidity of growth outside of the bone, we shall be forced to find the point of origin within rather than outside of the bone. It would be difficult to explain pictures such as are shown in Figs. 12 and 14 on any other basis. The erosion of substantia compacta occurs from within, as Figs. 15 and 17 illustrate; the periosteal spindle has its greatest width at the point of perforation; and the changes in the periosteum pursue the typical course described as belonging to intra-osteal growths.

Thirty-eight of the forty-six tumors under consideration, therefore, have their inception at points lying either in the spongy bone, in deepest layers of the compacta, or possibly in the marrow. My own series (as was already noted in a previous paper[15]) shows but one example of a neoplasm which is not central in origin. At that time my experience did not warrant any decisive conclusions either as to the question of the recognition of periosteal growths or as to

[15] Loc. cit.

the frequency of those that might be classed either as subperiosteal or peripheral. The additional facts gained in the analysis of Rumpel's specimens permits us to take the view that sarcomas do arise from the peripheral layers of the substantia compacta, usually in the shaft, and that Ribbert's assumption of a site of origin in the extremity of the long bones is untenable, at least so far as my own material is concerned. We encounter two types in the eight neoplasms before us. In the first there is a subperiosteal tumor without apparent destruction of bone, and in the second the tumor is associated with those osseous changes that are indicative of the neoplasm's destructive growth. Fig. 19, a diagrammatic illustration of one of Rumpel's cases, shows clearly the periosteal separation, the new bone formation without any appreciable lesion in the bone itself. Such a tumor, distinctly subperiosteal, must have its origin somewhere between periosteum and bone, and still not so intimately connected with either that recognizable dissolution takes place. Let us class these tumors then as of the subperiosteal variety.

When we find a broad sessile, or fusiform tumor on the shaft, with the signs of periosteal detachment, bone proliferation, and cortical erosion, we are led to refer the starting point to the outermost layers of the compacta. How else could we explain the pictures furnished in Figs. 18, 20, and 21. In each the shallow area of bone destruction seems to mark the beginnings of the growth. Fig. 20, perhaps, affords the most conclusive proof: for in the sagittal section we see clearly that the excavated focus posteriorly must have been the oldest lesion, lying as it does between the largest (and in their turn eroded) deposits of periosteal bone; spreading from this point and encompassing the shaft, it has produced elsewhere only the typical periosteal displacement and subperiosteal growth. The same history can be assumed for the neoplasm of the shaft of the femur depicted in Fig. 21.

In none of the specimens analyzed can we place the point of origin near the extremity of the long bone, and we are forced to the conclusion that the outermost layers of the corticalis should be regarded as the point of origin of the so-called peripheral forms.

Let us review briefly the grouping into 7 classes and inquire as to whether these will now stand the test of more careful investigation. Are the points of distinction that permit of this subdivision essential characteristics, are they purely fortuitous, do they depend wholly on the histological variations, or are they in great part but the expressions of differences in situation of the very beginnings of the growths? From what has already been said it is clear that the architecture of the sarcoma and the bone, as we see them, will not be fashioned by any one of those factors, but will show the effects of a combination of several or all. The utility of any grouping, it appears to me, must be estimated especially from the standpoint of value in diagnosis, and of value as an aid in remembering the types. A histological classification is practically useless in this regard.

The expansive types are sufficiently characterized by the absence of tendency to early perforation. The diffusely infiltrating growths may be said to remain confined to the interior of the bone because they arise in the deeper layers of the cortex, in the marrow, or in the middle of the spongiosa. That the site of origin must determine this peculiarity of remaining endosteal to a great extent cannot be denied. However, the late perforation, the slow invasion of the compacta, the extensive intramedullary involvement, the striking difference between the development here and in the dissolutive and perforative types—these facts warrant making a separate class of these forms. The fourth group—the dissolutive—presents a marked contrast to the expansive type, and merits therefore the special consideration which it has received. It might be urged that did this variety owe its inception to a focus near the cortex, the perforative endosteal tumor might result. We must grant this to be the case, and that this criticism is not a negligible one. We need hardly pause to discuss the utility of recognizing the sclerosing type; for the diagnostic points brought out by a study of the pathology of these tumors have been dwelt upon elsewhere. As regards the perforative endosteal tumors, can the gross structural changes be explained altogether by the assumption that here, too, the initial process is nearer the periphery than the centre of the bone? It seems that a difference in site of origin cannot be the only factor. For is it not striking that in the case of certain varieties (the diffusely infiltrating—and expansive) a marked resistance is offered by the cortical shell, whereas just here early penetration occurs? It would be only possible to explain the configuration of these tumors by supposing a beginning in the cortex itself. The relative size of extra- and intra-osteal masses will not permit of such an hypothesis in all cases. Those instances in which the extra-osteal tumor is not very much larger than the endosteal mass could hardly start so near the periphery, for the progress without must be much more rapid than within the bone. All in all, it seems best at this juncture to call attention to this variety by means of special grouping, leaving the final adjudication of the admissibility of such grouping until more specimens shall have come under observation.

Finally a word as to the peripheral forms. Ribbert includes here all those sarcomas that originate in bone of periosteal origin. Inasmuch as it becomes practically impossible to decide the exact point of origin from the study of the specimens, and a certain latitude of error must be allowed, such a basis for distinguishing the peripheral varieties seems inadequate. From a consideration of my own material and the study of the available data given by Rumpel, it would be best to include in the peripheral class only those neoplasms which show either subperiosteal growth without change in the bone, or subperiosteal growth with those typical erosive lesions that indicate to us a genesis in the most superficial layers of the cortex.

I wish to express my thanks to Dr. L. Jaches of the Mt. Sinai Hospital for some of the skiagrams utilized in this study, and my appreciation of the admirable work of Rumpel, which has added greatly to our knowledge of the diagnosis of bone sarcomas.

CASES ILLUSTRATING THE ORIGIN OF HYSTERICAL AND PSEUDOHYSTERICAL SYMPTOMS.

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The cases to be described will, it is hoped, elucidate a thesis which is rapidly gaining adherents, and which I have presented in various aspects in a series of articles upon hysteria.[1] In this is adopted the definition of Babinski that hysterical symptoms include all those, and are nothing but those "susceptible of being produced by suggestion and of being removed by suggestion-persuasion."[2]

The various corollaries and applications of this principle are developed at length in the aforesaid writings; and the value of the principle as a diagnostic criterion and guide to treatment has also been discussed, but will appear still more concretely in the following cases:

Case I.—*Electric Shock Palsy.* A girl was brought to Babinski, having become monoplegic upon receiving an electric shock while crossing a tramway line. This seemed like paralysis not caused by suggestion; but after the symptom had been removed by persuasion, further inquiry elicited the fact that the patient had overheard, some months previously, a conversation between some electricians, who were speaking of the dangers arising from electric shocks of the above description. It is evident that upon experiencing the shock there had flashed into the patient's mind a datum learnt from the conversation she had overheard and apparently forgotten, and that this memory furnished the suggestion at the base of the palsy she developed.[3]

[1] Considerations as to the Nature of Hysteria; Application to a Case, International Clinics, vol. iii, 1908; The Status of Hysteria, N. Y. Med. Jour., 1909, January 9; The Trend of the Clinical Concept of Hysteria, Bost. Med. and Serg. Jour., March 26, 1909; The Classification of Our Concepts of Hysteria, Month. Cyclop., March, 1909; The Essential Character of Hysteria New Orleans Med. Jour., June, 1909. The Genesis of Obsessions and Phobias and their Relation to Hysteria. Congress on Abnormal Children, New York, Medical Record, 1910.

[2] Ma Conception de l'Hysterie (conference devant l'Internat.), Paris, 1906.

[3] International Clinics, loc. cit.

It is perfectly clear that the paralysis of this girl existed merely on account of the idea that she could not move her limbs; that is to say, it was a mental (psychic) palsy. This idea was a false one, for, indeed, there was no interference with the neurons which performed limb movements; the idea had been implanted by the suggestion of a long-forgotten memory aroused into consciousness by an exciting stimulus. It was a true case of hysteria.

CASE II.—*Suggestions of Medical Origin.* So, also, was the case of traumatic neurosis cited by Brissaud,[4] in which the medical expert, Dupinet, who had previously examined the patient, and found him not anesthetic, saw an anesthesia suggested by the want of technical skill of another expert while examining the patient.

This case illustrates one of the origins of suggestions of medical source, faulty technique in examination of the sensibility of the patient, directly suggested that he could not feel.[5] The question "can you feel?" inevitably arouses the notion that it is possible not to feel, and this, in a state of mental preparedness, such as that of the apprehension aroused by medico-legal examination, affords a powerful suggestion. The evidence that such unskillful suggestions are frequently implanted by physicians who are indiscreet or unaware of the ease with which certain patients acquire false, fixed-ideas, will be found pretty fully presented elsewhere.[6] The gastric neuroses afford a fecund and illuminating example of this fact.[7]

CASE III[8]—*Typical Hysteria.* A servant girl, aged seventeen years, came to Ballet's clinic at the Hotel Dieu, Paris, on account of somnambulism, fits of anger, great fatiguability, loss of appetite, pains in the head, arms, and back, following overwork. After she was taken into his house by the writer, examination showed total insensibility to pin-prick on face, chest, or arms; but she felt deep pressure, and generally allocheirically. In the face, pin-prick was felt as a touch, and localization was uncertain. Cold appeared hot, except on the right hand, where it was indifferent, and on the head. Stereognosis was not impaired. Sensibility to touch was conserved on head and legs. Sense of attitudes only slightly impaired. Allocheiria on abdomen. Reflexes all normal, except that the right plantar was feeble, and the left conjunctival was absent. Watch heard, but imperfectly, especially on the left. Visual field restricted,

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[4] Discussion sur l'Hysterie, Revue neurologique, 1908.

[5] Le Rôle du Médecin en créant et en maintainent par ses Suggestions maladroites les Maladies produit par l'Imagination, Congrès de Lille, 1906, Amer. Med., August, 1908.

[6] The Traumatic Neurosis and Babinski's Conception of Hysteria. Congr. Internat. d'accid. Indust., Rome, May, 1909, and Medical Record, October 2, 1909. Also Jour. Abnor. Psychol., July, 1910; The Commonest Cause of Nervous Indigestion, Jour. Abnormal Psychol., February, 1909; Hypnotism Suggestion, Psychotherapic, 1891 and 1903; Les Fausse Gastropathes, Press méd., 1906; International Clinics, loc. cit.; Comment je comprends le Mot Hysterie, Bul. méd., 1906.

[7] The So-called Gastric Neuroses, Old Dom. Jour., Nov., 1908.

[8] International Clinics, loc. cit.